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CASE REPORT

Delayed cardiac tamponade following blunt thoracic trauma

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Introduction

Fatal cardiac tamponade may result from perforation of a cardiac chamber, major artery or vein. Delayed cardiac tamponade is a rare phenomenon that may appear days or weeks after the initial injury. This case describes cardiac tamponade occurring four days after a penetrating cardiac injury due to a flail segment.

Case report

An 18-year-old female was an unrestrained, front seat, passenger in a car involved in a head-on collision with a heavy goods vehicle. The car was estimated to be travelling at 110 km/h. The patient was ejected through the front wind-screen and was found by emergency services 30 m from the car.

Initial management was according to advanced trauma life support protocol. Clinical findings included an oxygen saturation level of 92%, a sinus tachycardia of 108 beats/min, blood pressure of 110/70 mmHg and a Glasgow Coma Scale (GCS) score of 15/15. Tenderness was noted over the left clavicle and ribs with crepitations audible throughout the left lung. The heart sounds were normal. Tenderness was also demonstrated over the lower lumbar spine and left sacrum.

Radiographs of the chest revealed left anterior rib fractures and pulmonary contusions. Cross-sectional imaging

with computed tomography (CT), obtained on presentation, demonstrated a left apical pneumothorax and multiple segmental fractures of the six upper left ribs producing a flail segment (Fig. 1). The fracture segments of the third and fourth ribs were angulated, compressing the left heart border. No haemopericardium was identified on CT and a portable transthoracic echocardiogram corroborated this. A chest drain was inserted, through the fifth left intercostal space in the mid-axillary line, draining 200 ml of blood.

Additional injuries included fractures of the left clavicle, scapula and transverse process of the third lumbar vertebra, a linear fracture through the left occipital bone and a stable left sacral fracture.

Multidisciplinary care was provided in the intensive care unit (ICU) and haemodynamic stability was maintained for the next three days. Patient-controlled analgesia and passive chest physiotherapy were provided. Ventilatory support was not required. Low-molecular-weight heparin was not administered. On the morning of fourth day in ICU, the patient deteriorated acutely with persistent hypotension and a sinus tachycardia despite intravenous fluid resuscitation. 500 ml of blood drained from the chest drain within minutes. The patient stabilised thereafter enough for a CT, which confirmed the presence of blood in the pericardial sac and in the mediastinum (Fig. 2). Backflow of contrast into the inferior vena cava corroborated a diagnosis of constrictive cardiac haemodynamics.

Ultrasound-guided pericardiocentesis was subsequently carried out and 400 ml of blood was aspirated. An immediate haemodynamic improvement was noted. Once stabilised,

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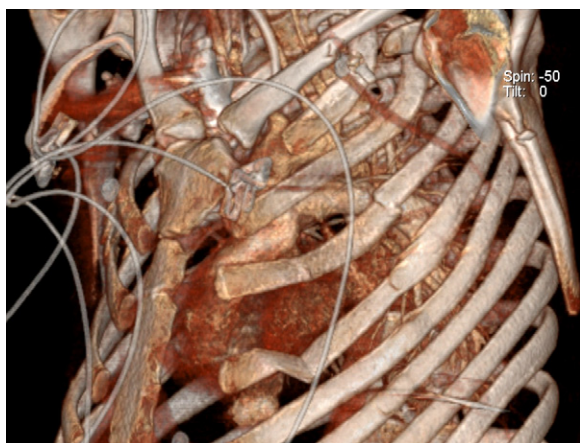


Figure 1 Three-dimensional reconstructed CT thorax demonstrating angulation of the fractured segments of the third and fourth ribs, compressing the left heart border. A displaced fracture of the left clavicle and fractured scapula are also noted.

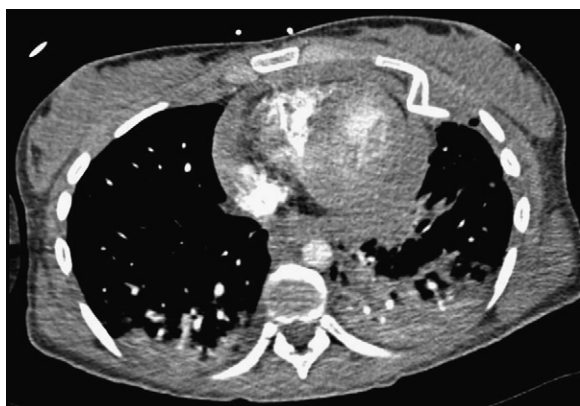


Figure 2 Axial CT thorax showing angulation of a fractured left fourth rib associated with haemopericardium and haemomediastinum.

arrangements were made to perform a median sternotomy semi-electively. Intra-operative findings included a 2 mm puncture wound in the pericardial sac (Fig. 3). Two further puncture wounds, 3 mm in depth, were noted in the left

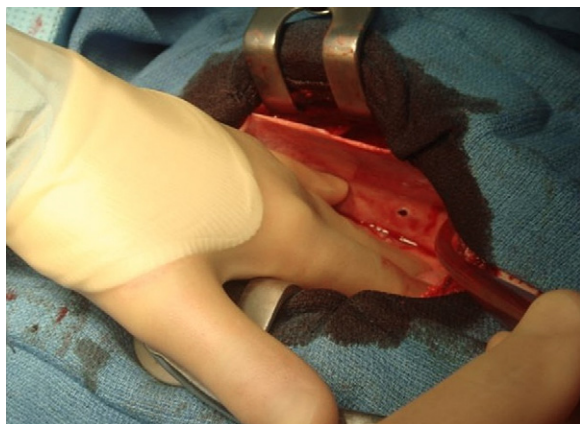


Figure 3 2 mm puncture wound in the pericardium caused by sharp rib fragments.

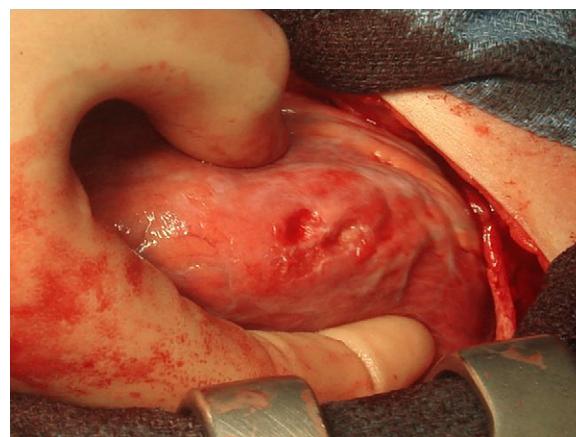


Figure 4 Puncture wounds, 3 mm in depth, in the left ventricular myocardium, the cause of cardiac tamponade.

ventricular myocardium (Fig. 4). The impinging rib segments were excised. The pericardium was closed. The post-operative course was uncomplicated.

Discussion

The majority of cases of traumatic cardiac tamponade occur following penetrating injuries; gunshots² and stab wounds¹ are the most frequently reported causative agents. Fewer than 20% of patients survive to hospital presentation⁷. Blunt trauma, as in our case, causing cardiac perforation and tamponade is unusual.

The exact mechanism whereby delayed cardiac tamponade occurs is unknown. Proposed theories include displacement of a thrombus that temporarily seals the cardiac perforation⁵ and torn adhesions formed at the time of the injury⁴. We suspect, in this case, that the ventricular injury occurred at the time of the road traffic accident. The puncture sites may have initially been sealed by clot, which were sub-radiographic. Subsequent dislodgement of the clot resulted in cardiac tamponade.

Physical findings are often non-specific and Beck's triad may go unnoticed in the setting of multi-system trauma. Multimodality radiological imaging is warranted, where the mechanism of injury raises the suspicion of cardiac injury. Transthoracic echocardiography is limited by severe chest wall injuries. Echocardiographic findings may be suboptimal as a result. Transesophageal echocardiography is beneficial in such cases³ but is not always available in timely fashion. CT or magnetic resonance imaging should be used if echocardiography is inconclusive⁸.

Isolated flail chest may be successfully managed with conservative measures such as pulmonary toilet and adequate analgesia. Intubation and ventilation is now reserved for patients with refractory respiratory failure. Surgical stabilisation is not routinely performed. Indications include severe chest wall instability, progressive decline in pulmonary function and when a thoracotomy is performed for other concomitant injuries⁶.

This case demonstrates the importance of maintaining a high index of suspicion in patients with blunt chest trauma. Potentially fatal cardiac tamponade may still occur despite an initial period of haemodynamic stability. There is no large

body of literature to specific risks or remedies and therefore management relies on first principles. Admission to the ICU is indicated with respect to respiratory status alone. The development of hypotension resistant to fluid administration, or disproportionate to the estimated blood loss should alert the clinician to the possible diagnosis of cardiogenic shock. Prompt diagnosis and definitive management, in this setting, can be life saving.

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